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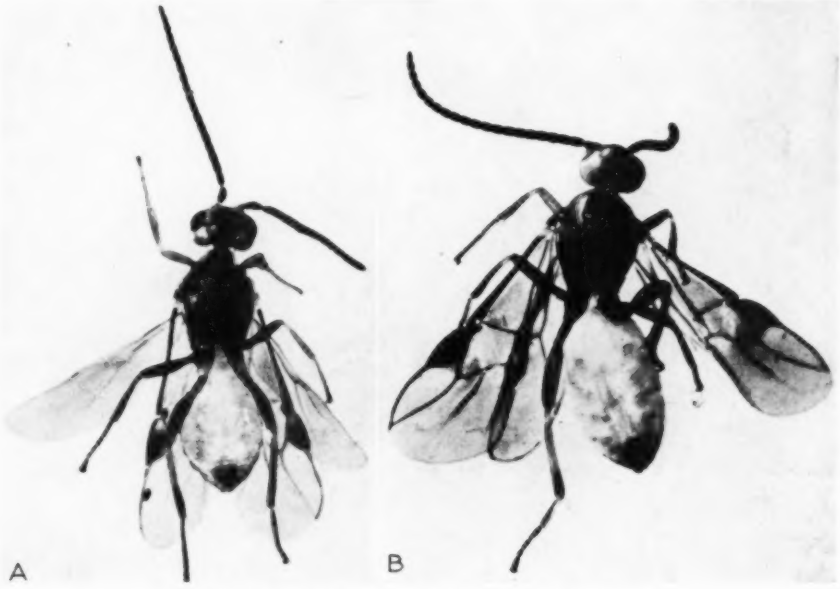
The Journal of HEREDITY



MOSAICS FOR FUSED-ANTENNA GENE

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Why Has the Tuberculosis Death-Rate Declined?

"Pruning-Shear" Parthenogenesis in Papaya

Sexual Photoperiodicity in Animals

Frequency of Multiple Births

The Silver Guinea Pig

VOLUME 26 JULY, 1935 NUMBER 7

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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The

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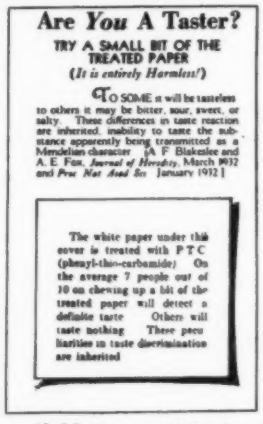
Are You A Taster?
TRY A SMALL BIT OF THE TREATED PAPER
(It is entirely Harmless!)
TO SOME it will be tasteless to others it may be bitter, sour, sweet, or salty. These differences in taste reaction are inherited, inability to taste the substance apparently being transmitted as a Mendelian character (A. F. Blakeslee and A. E. Fox, Journal of Heredity, March 1932 and Proc. Nat. Acad. Sci. January 1932)
The white paper under this cover is treated with P T C (phenyl-thio-carbamide). On the average 7 people out of 10 on chewing up a bit of the treated paper will detect a definite taste. Others will taste nothing. These peculiarities in taste discrimination are inherited

LEAFLETS TO DEMONSTRATE HEREDITY

Last year the "Taster test" served as an introduction to human genetic differences in many classrooms. The test is so easily made with the treated paper, and arouses such great interest that it appears to have a definite place in group demonstrations of such differences. These leaflets, size 3⅜" by 5½", are available in quantities for those who wish to use them for classroom or lecture purposes. On the back is printed an outline 4-generation pedigree chart. Sample on request. Prices: 10 for 25c; 50 for $1.00. 7" by 10" size, containing larger chart and more extended discussion of sense differences, 5 for 25c; 30 for $1.00.

AMERICAN GENETIC ASSOCIATION
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THE DERNER QUADRUPLETS

Frontispiece

These four daughters were born to Theodore Derner, Beuten, Germany, on December 19, 1927. Their names are Annelies, Maria, Victoria, and Etheltraut. At first glance the observer may doubt that they are identical. They seem to be separable into "two pairs of identical twins," but it is disconcerting that different people choose different pairs as being more alike. On closer feature-by-feature examination any such classification becomes increasingly difficult to maintain, and one suspects that a detailed study of resemblances and differences would demonstrate their origin from a single egg cell. In the United States at least two sets of quadruplets have lived to maturity in recent years. Curiously enough both these sets were all girls—the Gale sisters, apparently identical, and the Keys sisters, consisting of three identical triplets and a "sister."

A recent compilation of birth statistics in Germany for the years 1901-1925 has brought together some interesting information on the frequency of multiple births. The total number of births recorded during this period was 42,107,657. Of these births over half a million (531,541) were twins. The number of triplets (5,364) was almost exactly one per cent of the number of twins, and the number of quadruplet births (67) was again *approximately* one per cent of the triplet births. One set of quintuplets is recorded in these statistics covering a quarter of a century. In round figures this gives an average of one twin birth in 80, of one triplet birth in approximately 6,400 and one quadruplet birth in half a million. Quintuplets occur only once in about fifty million births, and instances of their survival for any length of time are phenomenally rare. The Dionne sisters who have held the stage of world interest and who are still all thriving at an age of fourteen months have long since broken all records for survival.

A formula has been presented by the German statisticians to express the frequency relation of multiple births: $1 : n^{x-1}$. In this n is the ratio of single births to twin births that occur in a given series of birth statistics (in this series $42{,}107.657 \div 531{,}541$), while x shows the "degree" of multiple birth in question (i.e. twins, triplets, quadruplets, etc.).

THE DECLINE OF TUBERCULOSIS

And the Cause or Causes of This Decline

ARTHUR R. DAVIAU

Health Officer, Waterville, Maine

EDITOR'S NOTE: *The questions raised in this article seem to be vital in formulating a "safe and sane" public health policy. Doctor Daviau's approach is frankly controversial and it is scarcely possible to cover so large a subject exhaustively in five pages. Unless valid reasons can be adduced to justify the belief that public health activities in New York, Chicago, and elsewhere should profoundly affect the tuberculosis death rate in Maine, Doctor Daviau's main thesis stands. If it is true that the dramatic fall in the tuberculosis death rate in recent years is due primarily to natural causes, it would seem to follow as an obvious corollary that the whole subject of communicable disease control should be given a searching reconsideration. If present public health theories result, as Doctor Daviau believes, "in incalculable suffering" it is clear that from the point of view of enlightened humanitarianism and economic expediency a revision of the methods of preventive medicine may be urgently required. How these diverse elements might be brought into harmony in recasting public health methods to avoid the difficulties envisioned by Doctor Daviau is a problem of great complexity, but one which if viewed broadly from a eugenic standpoint, might not be impossible of solution.*

THIS article is an attempt to prove that many public health workers accredit to their work end results which are the outcome of natural causes and are not the result of public health work.

Before I proceed with this article, I wish to state that I do not claim that to attain a higher natural immunity to various diseases we should allow everybody to be exposed to these diseases and then allow the amount of natural immunity to decide who shall live and who shall die. But if we wish to know where public health work is leading us to, we are forced to attempt a logical analysis of all the facts. If such an analysis proves that we are going in the right direction, we should keep on following our present public health methods; but if not, then, the sooner we modify our present methods the better for all concerned.

I can find no other disease in which the work of nature can better be demonstrated than in tuberculosis; and in no other diseases are public health workers more prone to accredit to themselves results which belong to natural causes alone.

The *Statistical Bulletin* of the Metropolitan Life Insurance Company, February, 1931, states:

> The campaign against tuberculosis has been successful far beyond what is indicated by the decline in the crude death-rate. The gains have benefited every group of the population, whether classified by sex, color, age, place of residence, or economic status. There has been nothing haphazard or accidental about what has happened. The results are a clear demonstration of the uncanny accuracy of the analysis made by the public health leaders 30 years ago as to the nature of the disease, its mode of transmission, and how best to check it. This may now be considered demonstrated in the history of the public health movement in America. . . . The drop in the tuberculosis death-rate has been most marked in the period since the influenza epidemic of 1918.

Then in the next issue of the same bulletin, one may read:

> The improvement has been much greater in the cities than in the rural districts. . . . The improvement shown for the cities is so much the larger that there can be no doubt that in our urban population a drop in mortality from tuberculosis has been greater than in rural districts.

In another issue of the same bulletin, September, 1932:

> It is in many ways a remarkable fact that, after two years of serious economic depression, the tuberculosis death-rate should continue its declining trend, for much historical and present day discussion has stressed the close relation between the tuberculosis death-rate and economic and social conditions. . . . It seems, therefore, that the forces for the control of tuberculosis among the thirty-four millions of people in the Original Registration States have been so well organized and coordinated as to

take care not only of the usual tuberculosis load, but also of accretions resulting from the economic depression. . . . Sanatoria are expanding their facilities. . . . Many thousands of lives are saved annually as a result of this effort alone. . . . Fewer deaths from tuberculosis mean that there are fewer advanced cases capable of producing new infections in the community.

The *Bulletin,* of the City of Chicago Municipal Tuberculosis Sanatorium, states:

The decline of tuberculosis seems universal, consistent, insistent. . . . Public health authorities and social agencies are perhaps inclined to take credit for the recent startling improvement in tuberculosis mortality and morbidity rates. Something has happened to tuberculosis outside our intent and volition. . . . The great drop from 1918 to 1922, is at present rather inexplicable to us. . . . Why this rapid decline? What is the explanation? We do not know. Influenza, increased standards of living, better educational methods, improved home hygiene, the sanatorium, all have been credited with a greater or lesser percentage of influence in this remarkable drop. As far back as statistics go we have never seen such a spectacular decrease in tuberculosis mortality. What is the explanation? Such a steep decline implies of necessity, some deep and consistent underlying motivation. We have not space in a pamphlet of this size to attempt to analyze the various factors mentioned, to try to assign their relative influence in the decrease. . . . We will merely take one of the factors mentioned—the sanatorium—and analyze it. What was its influence? We know with our present knowledge and the result of bitter experience that the sanatorium is not, in the usual sense, curative. . . . If anything, the cases under dispensary treatment alone showed a lower mortality rate than those who had sanatorium care.

In the course of his article, the author finally comes to the conclusion that sanatoria, thoracoplasty and municipal homes, due to the isolation of the carriers and the lessening of exposure, are the main known factors causing the decline in the tuberculosis death-rate in Chicago.

It is then seen that the authors of both bulletins come practically to the same conclusion, even though their deductions are based upon contradictory statements; but I am inclined to favor the Editor of the Chicago bulletin, for he at least admits that "Such a steep decline implies of necessity, some deep and consistent underlying motivation."

In the state of Maine, we lack adequate sanatoria facilities; thoracoplasty and other surgical methods have been practiced to a minimum, and we have no municipal homes; but nevertheless, *the tuberculosis mortality rate has dropped from 149.7 in 1910 to 49.6 in 1931.*

The chief factor causing the decline of the tuberculosis death-rate both in Maine and in Chicago, should logically be the same, but if we admit Chicago's claim, we then have to admit that respective local factors account for respective local declines, a postulate that seems neither rational nor logical.

If expensive preventive work is as efficient as is often claimed, Chicago's tuberculosis death-rate should show an uninterrupted yearly decline; but the death-rate was 56.8 in 1930 and 57 in 1931. We classify tuberculosis as a communicable disease and we state that the isolation of active cases has lessened exposure which has in turn resulted in a diminution of active cases; but without even attempting to correlate our statements, we often claim that 90 per cent of the population have had, have, or will have, tuberculosis, usually in a mild form.

In the *Public Health Reports* of Sept. 16, 1932, one may read, under the heading, "A Study of Tuberculosis among the Indians of Montana," by J. H. Crouch, M.D., C.P.H.:

On six Indian reservations in Montana, the T.B. death-rate per 100,000 population was as follows: Full-blooded Indians 1,357.7; mixed blood, one-quarter or more Indian, 339.1; mixed blood, less than one-quarter Indian, 52.3; White (in county), 23.7.

If many advanced cases mean greater exposure, and if more exposures imply more cases, then the whites in Dr. Crouch's survey should show a high tuberculosis incidence; but their death-rate for this disease is 3.7 lower than it is in Utah (which has a tuberculosis death-rate of 27.4—the lowest in a list of 27 states). Crouch's survey points clearly to the fact that resistance, either racial or individual, is the "consistent underlying motivation" that is univer-

sally, consistently and insistently reducing the tuberculosis death-rate. A number of considerations, such as the racial differences in susceptibility cited above, lead us to conclude that resistance is an inherited characteristic.

The Communicable Disease Cycle

Tuberculosis is a communicable disease, and as such should tend to follow the general course followed by other diseases of this nature. Unless we find a specific prophylaxis or a specific remedy against tuberculosis, (such as we have found for other diseases, such as small-pox, diphtheria, malaria, etc.), tuberculosis should tend to a minimum; a minimum reached by the elimination of those with the least resistance; a minimum acting as a barrier against the exposure of non-immunes. Lack of exposure should then permit non-immunes to live and to multiply until the immunes will no longer be numerous enough to create an effective barrier against the spread of tuberculosis; when the disease should again flare up in epidemic form. Following this outbreak it should again tend to the minimum point by the elimination of the non-immunes.

We have our recurring epidemics of mumps, measles, chicken-pox, etc.; these diseases flaring up in epidemic form whenever there have been brought into this world enough non-immunes to facilitate contact. This is well brought out in the case of the plague by Dr. Haggard in his book, "Devils, Drugs and Doctors," when he writes in effect: "Five years later the plague was again introduced into the city; but as the majority of the inhabitants were immune to the disease, this epidemic took a comparatively small toll of lives." We have plenty of data to prove that in many cities and states, the great number of those vaccinated against small-pox is actually acting as a barrier against the spread of the disease to the non-vaccinated.

All epidemics reduce the non-immune to a minimum only to be followed by the birth of more non-immunes, until the "concentration" of the latter in the population again reaches a point that makes an epidemic possible.

The reduction of the non-immunes to a minimum may be hastened by contributory factors. Thus influenza may have killed off many potentially tuberculous and thus have hastened the process of elimination. Whether due to the disease alone, or to other contributory factors, the end results are the same.

Although there is a great variation in the length of time taken by a communicable disease to start from epidemic form, reach a minimum number of cases and to again flare up in epidemic form, it would still be possible to estimate fairly accurately the length of such a cycle in some of the communicable diseases such as measles, chicken-pox, etc. Such a time factor would be more difficult to estimate in cycles of long duration as seems to be the case with tuberculosis. The time factor has no influence upon the logical claim that we do have communicable disease cycles; and if we do admit these cycles to exist, we are then forced to say that we are now witnessing the declining phase of the tuberculosis cycle.

Urban and Rural Rates

Tuberculosis is a disease of early adult life. In urban areas young adults marry older than in rural areas; often leaving no offspring before they are carried off by the disease. In rural areas, young adults marry younger, often have a few offspring by the time the diagnosis of tuberculosis is made, thus leaving a new potential tuberculosis crop.

According to the 1930 census, 58 women under 25 out of every 100 in the country are married. Only 47 in the same age class in the city are married. Among the boys, 31 in the country are married as compared to 25 in the city. A third more of the rural young people marry between the ages of 15 and 20 than do city residents of the same age.

Heredity, and not exposure, only can account for the slower tuberculosis death-rate decline in rural areas as compared to the urban areas.

To sum up, we may then say that

heredity in conjunction with the law of the survival of those who can survive, is the real factor accounting for the universal and gradual decline in the tuberculosis death-rate.

Effects of Faulty Premises

This article up to this point has been refused publication in a leading American journal of public health. Shortly after this there appeared an article on tuberculosis in a lay journal of wide circulation, the paper having been prepared by a prominent public health worker. The article began thus: "Until fifty years ago, tuberculosis was considered hereditary . . ."

We can hardly escape the conclusion that the public reaction to such a statement must have been racially and socially disastrous for one or more of the following reasons: (1) It gave tubercular people an incentive to multiply. (2) It deceived our welfare workers by strengthening their belief that assisting tuberculous persons to beget offspring was not only permissible, but rather laudable. (3) It made us overlook the ethical question involved in the right of tubercular persons to transmit their lack of immunity to the white plague to their offspring. (4) It predestined many unborn children to be born of physically defective parents, who due to disease are unable to take care of themselves, and thus still less able to care for their children. (5) It predestined many of these children to a life of poverty, often to be placed on the pauper lists of our towns and cities. (6) It predestined many a child to be born without the stamina and the prowess inherited by their luckier playmates. (7) It predestined many children to a life of medical care, with all its "does" and "dont's"; to the inconveniences of attending tuberculosis clinics; if not to be institutionalized for proper care, when the clinics failed to produce the *desired* (for we can hardly call them the *expected*) results. (8) If healthy, many of these children are nevertheless predestined to a life in an institution following the early death of parents suffering from the disease. (9) It predestined many unlucky human beings to a miserable, undesirable, pleasureless life, often only to be followed by a lingering death, occurring during what is supposed to be the most beautiful and enjoyable period of life, the teens and the twenties; for we must not forget that tuberculosis is primarily a disease of early adult life. (10) It encouraged the bringing into this world the bits of human wreckage, whom welfare workers endeavor to salvage at the expense and sacrifice of our healthy taxpayers, who are overburdened with more than enough taxes already. Such a statement deceives our citizens into the belief that more of their hard earned money is needed for the "humanitarian" work of bringing more derelicts into this world for us to endeavor to salvage, only to see them engulfed by the ocean of their own disease. Proofs of the above statements can be obtained from any sanatorium, pauper list or State Ward list.

Health Workers' Responsibility

It is true that this article may engender a controversy with public health workers, and that we are under no ethical obligation to enter into controversies when there is not much at stake, when we are dealing with a question of theory and not of facts. But when such a controversy concerns the welfare, the health, the well-being and the proper standard of living of thousands of human beings the need for plain speaking and frank discussion cannot be denied. When we look at tubercular patients not as "cases," but as hopeless and often defenseless suffering human beings; when the holocaust of thousands of wrecked lives must of necessity accompany the maintenance of an illogical idea, then it is our moral, humane, and I should say professional duty to question claims and interpretations that are not borne out by the facts. When we must try to find excuses to explain why the tuberculosis mortality rate has decreased during the depression, while according to what had been claimed before it should have risen; when we must, as I believe has been the case, improperly analyze statistics

to support a preconceived idea, failure to call attention to these inconsistencies can be due only to ignorance, indifference or cowardice. Had there been no controversies in the past we would still be in the Dark Ages. Nevertheless, I, as Health Officer, must admit that it is much easier, both mentally and physically, to ride with a popular idea than to try to fight it. Municipal homes, sanatoria, thoracoplasty and other public health measures, may make it possible for a human being to manifest whatever immunity he may have inherited to the disease; it may prevent exposure to the disease; but all these are only palliative public health measures. The increase of human beings with a natural immunity to tuberculosis seems to be the only curative measure which will absolutely and for all time rid our citizens of this scourge. It is true that reaching such an end might be practically impossible; but this fact should not prevent us from working towards that end, for all gain made in that direction will be a permanent gain, and not a temporary and highly speculative expedient.

I, as Health Officer, would like to bask in the glory that would be ours if the decline in the tuberculosis death-rate were due to our work alone; but such a glory is tainted. It has been stolen from the work that is being accomplished by a natural law, the law of the survival of those whose inheritance enables them to survive. If this is so we must admit that the pedestal on which our reputation rests is based upon almost incalculable suffering. If as a leader of the public in these matters I accept such praise its sweetness is marred by the thought that through my silence I am jointly responsible for the early death of many human beings who should never have been born, whose brief lives have been a burden, not only to themselves, and to their families, but indirectly to the entire community.

✦

ARTIFICIAL PARTHENOCARPY

JOHN H. SCHAFFNER*

THE tropical Pawpaw, *Carica papaya* L., is a dicœcious plant with a decided sexual dimorphism in both the inflorescence and the flower. The staminate flower is sympetalous with a slender tube while the carpellate flower has distinct petals and is in general much larger. It is well known that the staminate plant frequently undergoes sex reversal and becomes fruitful if the stems are split open or cut off. If the carpellate flowers are not pollinated, an abscission layer is promptly formed in the peduncle and the flower drops off. But if fertilization takes place an inhibitive process is initiated which prevents the production of this abscission and fruit development continues. This prevention of abscission by some influence or stimulus coming from the physiological processes originating during fertilization and the subsequent development of the embryo as well as the growth of the fruit itself is a typical case of ectogony, which may be defined as effect on nearby or surrounding parent tissues caused by the developing embryo or endosperm.

A number of years ago there was a solitary, vigorous carpellate plant of Carica in the botanical greenhouse of the Ohio State University. The plant bloomed abundantly but as there was no staminate plant to furnish pollen, fertilization could not take place and the flowers continued to develop abscission layers and dropped off. The writer thought it might be possible by some means to prevent the abscission by initiating a physiological condition similar to the inhibitive action, or ectogony, following fertilization. Accordingly all the large leaves were cut off and only a comparatively very small leaf surface was left on the plant. Thus the plant had a large, vigorous root

*Published as paper Number 361 from the Department of Botany, Ohio State University.

FRUIT GROWTH INDUCED WITHOUT POLLINATION
Figure 1

system taking in water with very little means for its escape. The formation of abscission layers was immediately stopped through the influence of the changed physiological gradients in the plant and the ovularies began a normal fruit development just as when the eggs in the ovules are normally fertilized. This was then an induced parthenocarpy which continued until the fruits were of considerable size (Figure 1). The writer went away during the summer and the young fruits were removed for preservation, since it was feared that they might be destroyed during his absence. The ovules remained entirely undeveloped and were minute structures on the surface of the fruit cavity.

The stimulus which caused the inhibition of the abscission and the development of seedless fruits could not have been of the nature of a hormone coming from a developing embryo and endosperm because no such structures were present. It is probable, therefore, that the various phenomena of ectogony are not due to migrating substances coming from a parasitic embryo but rather are of the nature of physiological disturbances which produce new gradients in the tissues and organs concerned. These disturbances may include electrical and radiation phenomena in relation to protoplasmic activity, of which we know very little at present. Even in the case of differential ectogony, as for example, in the date palm, where a developing hybrid embryo of a particular hereditary constitution may have a specific influence on the pericarp, there is no necessity for hypothecating migrating hormones until such are actually proven to exist. It is just as reasonable to postulate physical processes as chemical ones, at least until definite chemical relations have been established. It is evident that in the induced ectogony and subsequent parthenocarpy in the Papaya experiment, no hormone from a foreign source was available; and the unusual developments may have been caused entirely by stimuli produced by changed osmotic pressures because of the reduced leaf area through which the water coming from the roots could not escape.

SEX DETERMINATION IN BEES AND WASPS

P. W. WHITING*
Carnegie Institution of Washington, Cold Spring Harbor, N. Y.

THE Silesian priest Johannes Dzierzon was characterized by von Siebold as an outstanding bee-breeder and a keen and critical student. In 1845 Dzierzon put forth[5] the theory that drones (males) develop from unfertilized eggs while workers and queens (females) come from fertilized eggs. This theory was based on the facts that unmated and old queens produce drone broods and that race-crossing results in drones like the maternal race while the daughters are hybrid. Dzierzon's Law was long hotly contested and Dzierzon himself, in numerous publications appearing for more than thirty years, answered the arguments of his opponents.

It is also of interest to note that Dzierzon was aware of "mendelism" or segregating heredity twelve years before the publication of Mendel's paper on peas. In 1854 he stated[6] that the drones of the second generation from a cross resemble either the paternal or the maternal race and that these two types occur in equal numbers. He therefore glimpsed the fundamental gametic ratio.

Dzierzon's Law has been well established as the rule for the honeybee, although exceptions occur. The Cape Bee of South Africa (*Apis mellifera* var. *kaffra*), for example, produces females, both workers and queens, from unfertilized eggs laid by workers.[8]

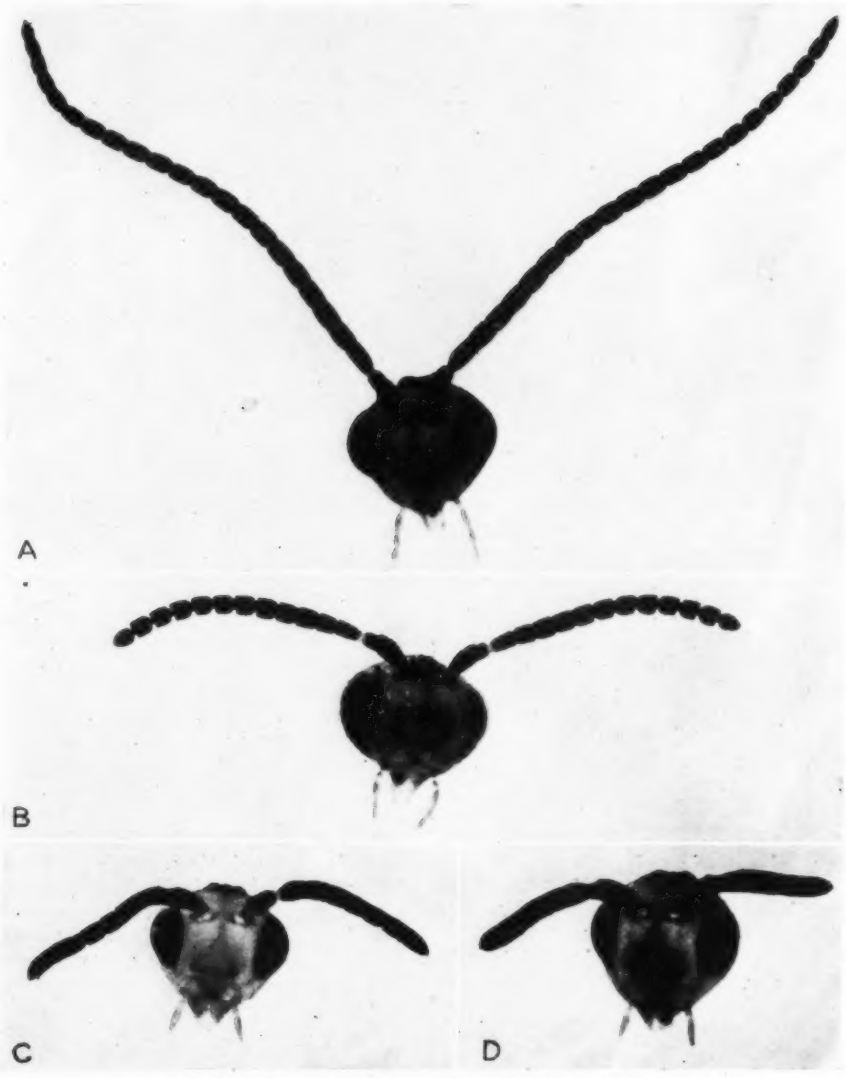
The law applies to other insects of the order Hymenoptera, including wasps, ants, ichneumon-flies, chalcis-flies and saw-flies, but many exceptions occur. In some species males are unknown and the females reproduce females indefinitely by parthenogenesis. Other species show alternation of generations, sexual males and females occurring at one season, parthenogenetic females (female-producers and male-producers) at another.

There appears to be no evidence that males are developed from fertilized eggs in any wild species of the order. In the honey-bee, however, biparental drones have been reported but the evidence is not entirely convincing. It is not improbable that they occur inasmuch as biparental males have been found in genetic work with the parasitic wasp Habrobracon.

Experimental and cytological work on Hymenoptera carried out since 1900 has led to the conclusions (1) that females are usually produced from fertilized eggs but may come from unfertilized: in any case they have at least the diploid number of chromosomes and (2) that in general males develop from unfertilized eggs and are therefore azygotes.

The azygote may be defined as an organism developing parthenogenetically from a haploid (reduced) nucleus. Cytological studies have shown that in such azygotes originating from haploid cells later cleavages may result in doubling of chromosome number so that the adult would be diploid and necessarily completely homozygous. Although the chromosome number characteristic of the male honeybee is sixteen as shown by Nachtsheim[10] in 1913 there is reason to suppose that this is double the haploid

*The writer is much indebted to the Committee on Effects of Radiation on Living Organisms (National Research Council) who have furnished grants for technical assistance for much of the research summarized in this paper. Financial aid has likewise been furnished from the Penrose Fund of the American Philosophical Society.



HEADS OF NORMAL AND MUTANT WASPS

Figure 2

Heads of wild-type male (*A*) and female (*B*). Note the sex difference in antenna length. Heads of male (*C*) and female (*D*) of the sex-linked mutant-type "fused" are shown for comparison. Note the extreme fusion of segments of antennae and of labial palpi as compared with wild-type. Sex difference in antenna length of fused mutants may also be noted, though this is not as great as in the normal.

set, since only eight tetrads are found in the first oöcyte. The male may then be a diploid azygote but some male tissues have a still higher number of chromosomes.

Genic Balance

It has been suggested that the principle of sex determination in the bee is similar to that in Drosophila since the male may be supposed to have one *X*-chromosome, the female two. The ratio of *X*-chromosomes to autosomes, however, remains the same in the two sexes of the bee, while in Drosophila this ratio is different, the females having relatively more *X*-chromosome material, the males having relatively more autosomal material.

The principle of genic balance postulates that certain genes tend to cause development in one general direction while other genes counteract this tendency. The character develops according to the resultant of these genic influences, but since each gene is represented several times in each cell and many times in the developing organism as a whole, the only constant relationship must be on a ratio basis rather than on the basis of an algebraic sum. Hence as applied to sex determination in the bee, the theory that the female has merely the equivalent of double the male set of chromosomes (or genes) is not in agreement with principles held for other forms.

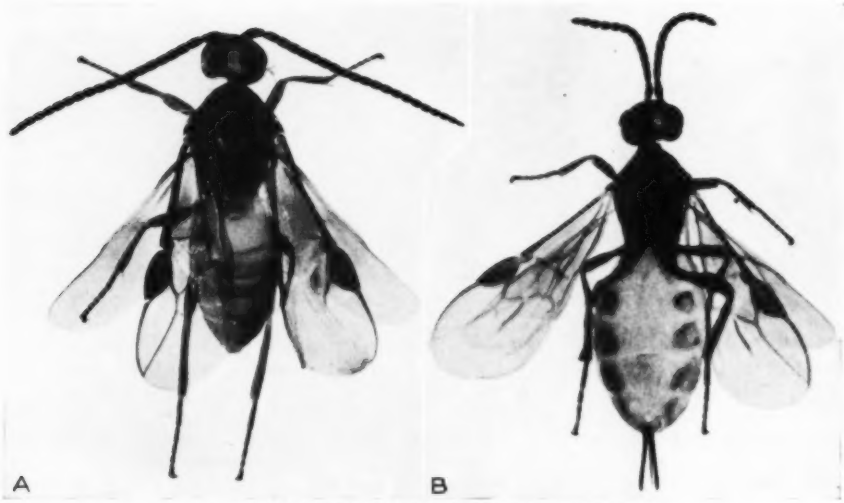
Mendelism and Sex Determination

In the male honey-bee (drone) the first meiotic division does not involve the nucleus. There is merely a small cytoplasmic bud or "polar body" given off. The second division appears to be equal as regards the nucleus, but practically all the cytoplasm remains at one pole. The smaller cell or "second polar body" degenerates and only one sperm cell is formed from a spermatocyte. Petrunkewitsch[11] concluded after embryological study that while the body of the male bee is haploid, the gonads are diploid and derived from a fusion of two polar nuclei after maturation of the egg. Nachtscheim[10] has shown this conclusion to be erroneous.

Castle[4] in 1903 was the first to apply the mendelian principle of segregation to sex determination in the bee. He postulated differential maturation not only for the egg but also for what he supposed, following Petrunkewitsch, to be a reductional division of a diploid spermatocyte. A pair of allelomorphic factors, maleness and femaleness, are concerned, femaleness being dominant. The female is heterozygous, but femaleness always passes into the polar body so that the unfertilized egg develops into a haploid male. The testes, supposed to originate from a polar fusion nucleus, are diploid and heterozygous for sex. Castle supposed maleness to pass into the polar body in the maturation of the sperm, while dominant femaleness remains in the sperm so that all fertilized eggs develop into females.

Nachtsheim[10] suggests that the ancestral Hymenoptera may have been diploid and digametic in the male but that when parthenogenesis and male haploidy were acquired the first spermatocyte division became abortive so that no male-producing spermatozoa are developed. He pointed out that the second spermatocyte division is equational with respect to the chromosomes, as it is in the ants and wasps in which the cytoplasm, unlike that of the bee, divides equally. Nachtsheim therefore concluded that the haploid set of chromosomes determines maleness, the diploid set femaleness. He searched for evidence of female digametism but failed to find any constant difference indicating *X* and *Y*. He suggested* differential

*"Es war ja denkbar, dass ein solches im Ei vordhanden wäre und vielleicht in dem befruchteten und unbefruchteten Eiern verschieden suf die vier Chromatingruppen verteilt würde." (Nachtsheim 1913, p. 187.)



A MOSAIC MALE AND NORMAL FEMALE

Figure 3

A—Male mosaic for the mutant trait veinless. The difference may be seen in the primary wings—the side to the right having few veins in the wings. Both secondaries are veinless. *B*—Ventral view of wild-type female.

maturation of the egg directed by presence or absence of the sperm nucleus. This is closely comparable with the scheme advanced by Castle except that it is free from Petrunkewitsch's errors in regard to the origin and genetic composition of the male gonad.

This suggestion of Nachtsheim, as well as Castle's hypothesis, although erroneous in certain details, is more consistent with modern ideas of genic balance than is the view finally adopted by Nachtsheim, and presented by him in more recent publications, that the chromosome composition of the female is merely double that of the male.

Sex Types in Habrobracon

The problem of sex determination in the Hymenoptera and in other forms with azygotic males will be better understood after consideration of the principles that have been disclosed by means of genetic and cytological work with the parasitic wasp *Habrobracon juglandis* (Ashmead).

Habrobracon produces normal haploid males from unfertilized eggs and normal diploid females from fertilized eggs. Occasionally a normal diploid female is produced by a virgin mother from crosses of certain stocks[12].

A gynandromorph may be produced from a binucleate egg if one of the nuclei is fertilized. Male parts of the body are therefore matroclinous, female parts biparental.

If the parents are closely related diploid biparental males occur in relatively small numbers, the ratios differing according to stocks crossed[1]. These diploid males show no evidence of feminization either in external or in internal structure.

Occasionally a haploid mosaic male develops from an unfertilized egg laid by a female heterozygous for one or more genes. These mosaic males show in different parts of the body the alternative traits for which the mother was heterozygous. Thus the eyes which are black in the wild-type may be in part orange if the mother was heterozygous for orange. In case the mother was heterozygous for two nonallelomorphic genes affecting the eyes,

ivory, o^i, and white, *wh*, for example, a mosaic son may be ivory in part of the eye, white in another part. In this case no wild-type tissue occurs. Nevertheless in the ivory region adjoining the white area there will be found black pigment which gradually fades out as the distance from the white increases. No black pigment is found in the white region. It appears that some diffusable substance had been produced in the white region by the dominant allelomorph to ivory. This substance coming in contact with tissue containing the dominant allelomorph to white interacts in such a way that black pigment resembling that of the double dominant wild-type is developed[13]. A rather high proportion of these mosaic males show feminized structures in the genitalia and more rarely in other parts also (Figure 6*C*)[17]. On the basis of comparison with the eye color effect the hypothesis was suggested[14,15] that these feminized mosaic males are mosaic for at least two sex factors. One type of tissue contains *F.g* (in the *X*-chromosome), the other contains the allelomorphs *f.G* (in the *Y*-chromosome). Either recessive factor causes maleness, but *G* produces some diffusable substance which, coming in contact with tissue containing *F*, interacts in such a way that feminization results. The diffusion is apparently in one direction only as it is in eye-color.

Two kinds of males were postulated, *F.g* (or *X*) and *f.G* (or *Y*), which have been identified genetically, although they are phenotypically similar. The female contains both the *X*- and the *Y*-chromosomes and is therefore heterozygous or digametic *F.g/f.G* (or *X/Y*). The dominant factors present in the two types of males are complementary to each other in producing femaleness. Males normally have one set of autosomes ($1A$) while females have two sets ($2A$). A female produces from unfertilized eggs $1X + 1A$ and $1Y + 1A$ males in equal numbers. If crossed with a $1Y + 1A$ male she might be expected to produce from fertilized eggs, females $1X + 1Y + 2A$, and diploid

Table 1. Chromosomal types in Habrobracon.*

Sex			Chromosomes	Genes
Males. Impaternate. (Haploid)		Type 1	$\frac{1X^{+1}A}{}$	$\frac{F.g}{}$
		Type 2	$\frac{1Y^{+1}A}{}$	$\frac{f.G}{}$
Biparental (or impaternate). (Diploid)		Type 1	$\frac{2X^{+2}A}{}$	$\frac{F.g/F.g}{}$
		Type 2	$\frac{2Y^{+2}A}{}$	$\frac{f.G/f.G}{}$
Sons of biparental males. (Triploid)		Type 1	$\frac{3X^{+3}A}{}$	$\frac{F.g/F.g/F.g}{}$
		Type 2	$\frac{3Y^{+3}A}{}$	$\frac{f.G/f.G/f.G}{}$
Mosaic. (Haploid)		Type 1	$\frac{1X^{+1}A}{1X^{+1}A}$	$\frac{F.g}{F.g}$
		Type 2	$\frac{1Y^{+1}A}{1Y^{+1}A}$	$\frac{f.G}{f.G}$
		Type 3	$\frac{1X^{+1}A}{1Y^{+1}A}$	$\frac{F.g}{f.G}$
(Haplo-diploid)		Type 1	$\frac{1X^{+1}A}{2Y^{+2}A}$	$\frac{F.g}{f.G/f.G}$
		Type 2	$\frac{1Y^{+1}A}{2Y^{+2}A}$	$\frac{f.G}{f.G/f.G}$
		Type 3	$\frac{1X^{+1}A}{2Y^{+2}A}$	$\frac{F.g}{f.G/f.G}$
		Type 4	$\frac{1Y^{+1}A}{2X^{+2}A}$	$\frac{f.G}{F.g/F.g}$
Females. Biparental (or impaternate). (Diploid)			$1X^{+1}Y^{+2}A$	$F.g/F.G$
Daughters of biparental males. (Triploid)		Type 1	$1X^{+1}Y^{+2}A$	$F.g/F.g/f.G$
		Type 2	$1X^{+1}Y^{+2}A$	$F.g/f.G/f.G$
Gynandromorphs. (Haplo-diploid)		Type 1	$\frac{1X^{+1}Y^{+2}A}{1X^{+1}A}$	$\frac{F.g/f.G}{F.g}$
		Type 2	$\frac{1X^{+1}Y^{+2}A}{1Y^{+1}A}$	$\frac{F.g/f.G}{f.G}$
(Diploid)		Type 1	$\frac{1X^{+1}Y^{+2}A}{2X^{+2}A}$	$\frac{F.g/f.G}{F.g/F.g}$
		Type 2	$\frac{1X^{+1}Y^{+2}A}{2Y^{+2}A}$	$\frac{F.g/f.G}{f.G/f.G}$

*The haploid set of chromosomes includes in addition to one set of autosomes ($1A$), an *X*-chromosome ($1X$) or a *Y*-chromosome ($1Y$). The chromosomes of a normal haploid male are one or the other of these haploid sets. The chromosomes of the other types are some multiple or combination of these sets. Aneuploid types have not been found except for a few cases of mosaicism by elimination not included here. The *X*-chromosome contains the two genes or two groups of genes *F.g*. The *Y*-chromosome contains the allelomorphs *f.G*. Homologous genes in the same cell (diploidy, triploidy) are separated by / in the genic formulae. Tissues of different genetic constitution or of diverse origin (mosaicism) are separated by horizontal lines in the chromosomal or in the genic formulae.

Biparental males are regularly obtained when parents are related. Impaternate diploid males are to be expected among the brothers of impaternate females but have not yet been identified. A very few (triploid) sons of biparental males have been obtained.

Haploid mosaic males are presumably of three types but only Type 3 is mosaic for sex. This type frequently has feminized genitalia (gynandroidism). A few haplo-diploid male mosaics have been found, the haploid parts matroclinous, the diploid parts biparental. The four types are presumptive.

Diploid females are regularly biparental but impaternates occur in small numbers when certain stocks are used. Daughters (triploid) of biparental males occur in small numbers. The unbalance between *X* and *Y* may account for their degenerate ovaries and other morphological abnormalities.

Besides the gynandromorphs with matroclinous male parts and biparental female parts (haplo-diploid), a very few have been found in which male parts also are biparental (diploid).

males, $2Y+2A$, in equal numbers. Similarly if a female is crossed with a $1X+1A$ male, the diploid sons should be $2X+2A$. It may be seen from these formulae that the genic ratio of X to A or of Y to A is the same in the diploid males as in the corresponding haploid, while the female is unlike either, being a combination of the two. Moreover females are necessarily diploid for they must have both dominant factors F and G which are carried in separate but homologous chromosomes.

Tests for Sex-Linkage

Proof of this hypothesis of sex determination was to be sought by means of a sex-linked factor. If we designate the factor to be tested as a and its wild-type allelomorph as A, a recessive male must be either aX or aY and a heterozygous female must be either AX/aY or AY/aX. The four possible types of matings with results expected in case of complete sex-linkage are shown in Table II. Impaternate males should be wild-type and mutant-type in equal numbers whether the factor in question is sex-linked or not. If the factor is not sex-linked wild-type and mutant-type should occur in equal numbers in both sexes. If the factor is sex-linked the biparental sons should be of the type opposite to that of the daughters. If a recessive male be crossed to his own daughters, his second generation daughters will be wild-type. Crosses 1 and 3, Table II, are examples of such crosses since the recessive factor will be Y-linked if the father is aY or it will be X-linked if the father is aX. Since biparental males cannot be distinguished from impaternate males, unless the mothers are homozygous for a factor for which the father is dominant they must be disregarded in these backcrosses. If crossing over occurs between the factor in question and the XY region of the chromosome, both types of females and of biparental males will be expected and the ratio of the crossover type should be equal in the females and in the biparental males. Since X and Y males cannot be separated by inspection, a convenient test for sex-linkage is to cross a single recessive male with the heterozygous daughters of a single recessive male. Thus large numbers may be obtained from many cultures of similar type.

Very few crosses of heterozygous females to recessive males had been made in previous work with Habrobracon. The original mutant to orange had been crossed with his heterozygous daughters in order to obtain orange females. These occurred in excess of their heterozygous sibs which was opposite to expectation in case of sex-linkage. Although numbers were small it may be concluded therefore that orange is not sex-linked. Eleven other genes were tested in 1933 (Table III). Ratio of wild-type to mutant-type in the females may be compared with that in the males. If there is no sex-linkage, equality is expected in both sexes, except for differences due to viability. If the mutant-type is less viable than wild-type the deficiency may be in the same direction in both sexes,

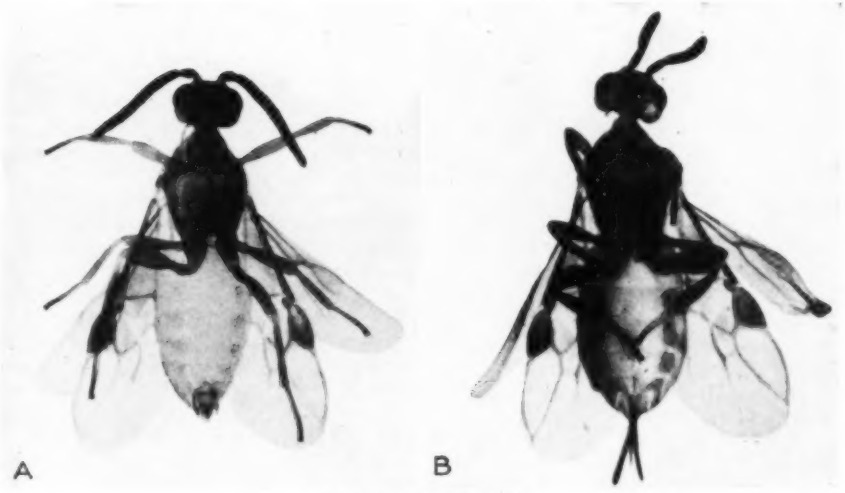
Table II. Expectation in case of complete sex-linkage for the four possible crosses of heterozygous females by recessive males.

Crosses	Parents		Offspring			
			Impaternate		Biparental	
	♀♀	♂♂	♂♂		♀♀	♂♂
1.	AX/aY	aY	AX	aY	AX/aY	aY/aY
2.	AX/aY	aX	AX	aY	aY/aX	AX/aX
3.	AY/aX	aX	AY	aX	AY/aX	aX/aX
4.	AY/aX	aY	AY	aX	aX/aY	AY/aY

Table III. Tests for sex-linkage of various genes.*

Groups.	Genes involved	Offspring			
		♂♂		♀♀	
		wild-type	mutant-type	wild-type	mutant-type
1.	orange, o (eyes).	221	243	44	59
2.	maroon, m (eyes).	21	19	21	19
3.	white, w (eyes).	131	144	140	143
4.	eyeless, el.	490	420	109	120
5.	crescent, cr (eyes).	72	46	20	28
6.	tapering, ta (antennae).	59	63	34	38
7.	wary, wa (wings).	419	410	156	140
8.	shot-veins, sv (wings).	117	130	135	150
9.	indented, in (wings).	142	139	138	94
10.	small-wings, sw.	150	98	59	71
11.	cut, ct (wings).	119	107	88	54
12.	cut, ct (wings).	49	57	111	113
13.	cut, ct (wings).	150	148	165	165
14.	cantaloup, c (eyes).	148	112	294	299

*Offspring produced by heterozygous daughters of single recessive males either backcrossed to their recessive fathers (Groups 1-11) or to one other recessive male (Groups 12-14). In case any gene is sex-linked, it is to be expected that one class among the females will show a marked divergence from the same class among the males. If the backcross is made there should be a deficiency of the mutant-type females as compared with the mutant-type males. Biparental males could not be distinguished from impaternate since mothers had no recessive factor difference from fathers. There is no evidence that any of these genes are sex-linked.



"FUSED" MUTANTS

Figure 4

Ventral views of fused male (*A*) and female (*B*). Sex difference in genitalia and in size of abdominal sternites as also in antennal length may be noted. Fusion of segments of tarsi as well as of antennae and labial palpi is characteristic of this mutant-type. There is also reduction in size of the wings. The primaries have a slight indentation in the costal margin near the tip.

so that a deficiency of mutant-type females does not indicate sex-linkage if mutant-type males are also deficient. There is obviously no close sex-linkage for any of the genes listed in Table III.

Sex-Linkage of Fused

Before these tests were completed it was recalled that in 1930 Neita C. Bostian had reported a very irregular fraternity involving the recessive mutant factor fused, *fu*. This mutant-type found by C. H. Bostian in 1929 has extreme fusion of segments of antennae, of tarsi and of labial palpi, and a slight notch at the end of the vein along the costal margin of the primary wings (Figures 2*C*, *D* and 4). Fused females are sterile and it is often difficult to obtain matings of the males, but when matings do occur a good proportion of the eggs are fertilized. Fused segregates sharply from wildtype. Five recurrences of the mutation have been reported.

The fraternity reported by Neita C. Bostian was from a heterozygous mother and a recessive father. Sons were wild-type and fused in approximately equal numbers as expected but all daughters were fused. No explanation could be given at the time, but this case now suggested that fused might be sex-linked. Sex-linkage was probably not complete for crossing over must have occurred in order to get a combination that would give mutant-type females (Crosses 2 and 4, Table II).

Tests for sex-linkage of fused were made by B. R. Speicher in autumn of 1933 (Table IV, Groups 1-8). A certain fused male, designated *fuX#1* was crossed to a homozygous wild-type female (stock 25). Two of the daughters, *Y/fuX#1*, (the symbol for the wild-type allelomorph to fused is omitted) were backcrossed to the father (Group 1). Four other daughters (Group 2) and five later generation females (Group 3) with fused-*X* fathers were crossed with other fused males. A marked excess of wild-type daughters was produced in each case. Six other daughters

of the original *fuX*#1 were crossed with fused males and gave a marked excess of fused daughters (Groups 4 and 5). These males were therefore fused-*Y*. Other crosses of these fused-*Y* males with daughters of the original *fuX*#1 gave near equality of wild-type and fused among the daughters (Groups 6 and 7) and one daughter of one of these fused-*Y* males crossed with a male known to be fused-*X* likewise gave equality (Group 8). It is clear that there are three kinds of fraternities distinctly different as regards percentage of fused among the females, showing either great deficiency, great excess or near equality. It also appeared that there are too kinds of males and that no one male sires both excess and deficiency of fused daughters in different fraternities from females which are sired by the same male. However, the same male may sire excess of fused and equality of fused in different fraternities from daughters of the same male. (Compare Groups 4 and 5 with Groups 6 and 7 respectively). The facts are all in agreement with the hypothesis of close sex-linkage of fused, but some females heterozygous for fused show independent assortment among their progeny. It is suggested that a chromosome translocation may be the explanation in these cases where linkage is lacking.

As stated above the biparental males may be separated from the impaternate only if the father has a dominant factor which is lacking in the mother. Reëxamination of certain data previously collected by Magnhild Torvik-Greb was made and it was found that here also the fraternities fell into three different classes as in the tests made by B. R. Speicher. The heterozygous mothers were orange-eyed and the fused fathers were black-eyed so that it was possible to separate the biparental males from the impaternate. The few biparental males were produced and even these were not all classified with respect to fused. It is suggestive, however, that those fraternities showing a marked deficiency of fused among the females had an excess of fused among the biparental males (Table IV, Group 9) and the reverse (Group 10). There were also four fraternities with near equality of fused and wild-type among the females (Group 11). These contained no biparental males, although at least one biparental male occurred in each of the fraternities showing linkage of fused and *XY*.

Recently C. H. Bostian[2] has published summaries of his experiments in which orange-eyed females heterozygous for fused were crossed with very closely related black-eyed fused males (Table IV, Groups, 12, 13). Biparental males were produced in large enough numbers to prove definitely that they occur in proportions with respect to fused that are opposite to those of their sisters. The material was checked for the *X*- and *Y*-chromosomes by a breeding test with that of B. R. Speicher.

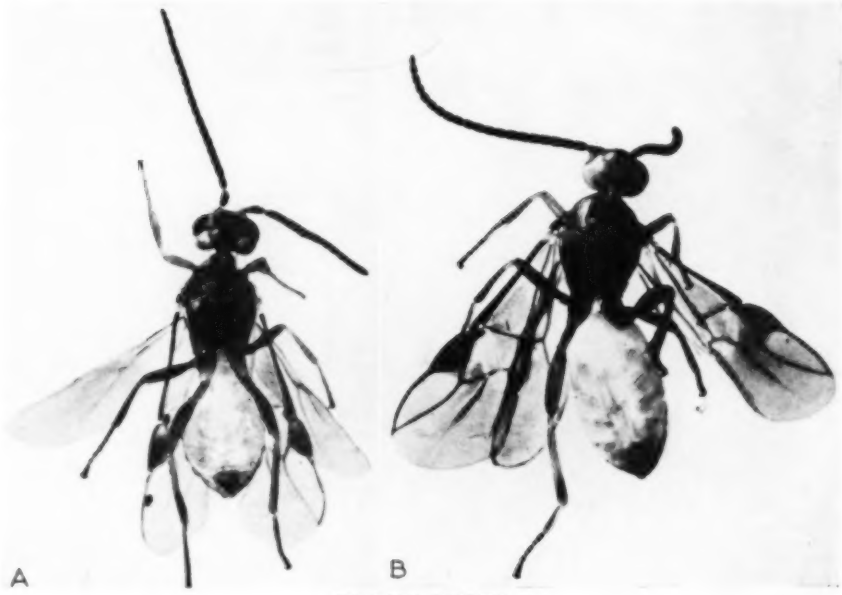
Biparental Males and Selective Mortality

Biparental males are always much less frequent than females. They are

Table IV. Tests for sex-linkage of fused. *

Fraternities		Parents		Offspring					
		♀♀	♂♂	♀♀	♂♂				
					black			orange	
Groups	No.			+	fu	+	fu	+	fu
1.	2	Y/fuX#1	fuX#1	14	1	5	4		
2.	4	Y/fuX#1	fuX	108	6	42	45		
3.	5	Y/fuX	fuX	76	11	26	40		
4.	4	Y/fuX#1	fuY#5	23	226	29	77		
5.	2	Y/fuX#1	fuY#4	11	122	27	48		
6.	2	Y/fuX#1	fuY#3	56	74	25	17		
7.	3	Y/fuX#1	fuY#4	78	68	18	14		
8.	1	X/fuY#4	fuX	18	18	4	8		
9.	7	o/o +/fu	fu	180	8	2	7	6	112 180
10.	8	o/o +/fu	fu	9	68	2	1	1	88 98
11.	4	o/o +/fu	fu	71	60				87 92
12.	13	o/o +/fuI	fuI	96	905	92	16		220 197
13.	40	o/o +/fuI	fuI	1975	195	41	230		577 510

* Data from crosses of females heterozygous for fused by fused males. In Groups 1-8 mothers were black-eyed and sons can therefore not be separated into biparental (black) and impaternate (orange) as they can in Groups 9-13. Fused may be either in the *X*-(*fuX*) or in the *Y*-(*fuY*) chromosome. Individual fathers are designated as #1, #3, #4 and these numbers entering into the formulae of the mothers indicate the source of their *fuX* or *fuY* chromosome. Thus Group I includes two matings of daughters (*Y*/*fuX*#1) back to their father (*fuX*#1). Fraternities fell into three distinct classes with respect to percentage of fused among the females, which may be very low, 10.3% (Groups 1, 2, 3, 9, 13), very high, 86.5% (Groups 4, 5, 10, 12) or near equality, 48.5% (Groups 6, 7, 8, 11). The first two classes indicate linkage with the factors for sex or the *XY* region of the chromosome: the last class suggest that a chromosome break and a translocation has occurred.



MOSAIC MALES

Figure 5

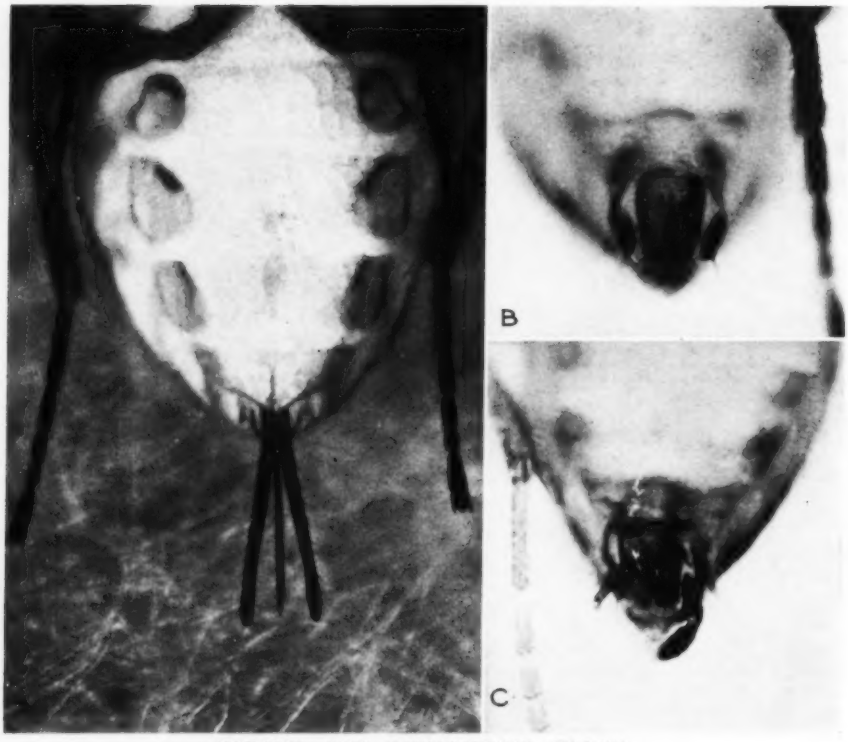
A—Male mosaic for veinless (wings) and for fused (antennae, tarsi, wings). The left antenna probably contains both fused and non-fused tissue. The left wings are fused non-veinless, the right veinless non-fused. *B*—Male mosaic for fused (antennae, wings, tarsi) and for orange (eye color).

totally lacking when parents are unrelated. If parents are related biparental males may comprise less than one per cent of the total biparental offspring or they may range as high as twenty-five per cent or more from crosses of certain stocks as shown by C. H. Bostian[1]. They have, however, never equalled the females as expected on a mendelian basis. What is the explanation of this?

For several years it had been evident that females mated to closely related males produce fewer offspring than their virgin sisters and that such mated females show an increase in fecundity when they cease to produce daughters due to exhaustion of sperm supply.

Hase[7] counted the eggs and offspring from three mated females. Female *A* produced 243 inviable (188 eggs, 55 larvae or pupae) and 74 viable (43 males, 31 females) or 23 per cent viable. Female *B* produced 126 inviable (98 eggs, 28 larvae or pupae) and 132 viable (129 males, 3 females) or 51 per cent viable. Female *C* produced 135 inviable (119 eggs, 16 larvae or pupae) and 66 viable (34 males, 32 females) or 33 per cent viable. It should be noted that viability was highest in the fraternity including few females (from Female *B*). The other fraternities, having many eggs fertilized, may have been reduced in size by the inviability of biparental males. It is not unlikely that the mate of Female *B* was himself a biparental, siring but three (triploid) daughters.

An experiment carried out by Anna R. Whiting in 1923[16] demonstrated that fertility of eggs from outcrossed females is equal to that from virgin females but is much reduced by fertilization with sperm from males of the same stock. Seven virgin females laid 2,068 eggs, 59 per cent of which produced adults (all males). Two outcrossed females laid 374 eggs, 58 per cent of which produced adults (81 per cent females). Two females mated with males of the same stock



NORMAL AND GYNANDROID FORMS

Figure 6

Ventral views of external genitalia of normal female (*A*), normal male (*B*), and of gynandroid male (*C*). Gynandroid males have in addition to the male set of external genitalia, one or more reproductive appendages resembling a short ovipositor sheath or even a sting. These are produced laterad to the male outer claspers. The specimen shown at *C* has a relatively large left ovipositor sheath, a very small right one. The gynandroid male is a mosaic of *X* and *Y* tissue. The female-like structures developed in the genitalia are phenotypic and due to a complementary action between the two different types of male tissues.

laid 580 eggs, only 40 per cent of which produced adults (50 per cent females). Reduction in fertility associated with lowering of female ratio by inbreeding is due to production of relatively inviable biparental males. Two females crossed with biparental males laid 480 eggs with 63 per cent viability. Two inviable and eight viable but sterile triploid females occurred. This fraternity may be compared with that from Female *B* in Hase's experiment.

In an experiment carried out in 1925-26 with closely inbred stock, Arthur M. Cloudman[16] showed negative correlation between ratio of females and number of offspring produced. If many eggs were fertilized by closely related sperm, many homeosyngamic combinations, *XX* and *YY* (biparental males), would be formed as well as heterosyngamic *XY* (females). The inviability of the homeosyngamic combinations would tend to increase the female ratio and lower total numbers in the fraternities involved.

Selective mortality is at least a partial explanation of the facts and it is possible that different stocks carry factors which

are more or less lethal to biparental (diploid) males, but which have little or no deleterious effect on normal (haploid) males or on (diploid) females.

This, however, is not the whole story. The total absence of biparental males among many thousands of biparental offspring (uniformly females) from crosses of unrelated stocks is difficult to explain on this basis. Moreover there is much greater fecundity if females are outcrossed than if they are mated to closely related males. Were selective mortality the only factor involved we would expect fecundity of females to be increased with increase in ratio of males among the biparentals. Correlation of fecundity with ratio of males among the biparentals should be positive, where as it is actually negative.

It has also been shown by Anna R. Whiting[16] that among progeny from various crosses of different inbred stocks, ratio of biparentals varies inversely to ratio of males among the biparentals and it has been shown by Torvik and Wenstrup[1] that among fraternities produced by inbred females mated to segregating males (from crosses of stock related by stock unrelated to the females) there is negative correlation between these two ratios. Biparental offspring, therefore, are more frequent if they are all females.

Sex Reversal

Evidently we need an hypothesis supplementary to differential viability. It has been suggested that sex-reversal may be the explanation of the biparental males, that they are females turned into males by some condition resulting from inbreeding or close relationship of parents. This theory is not in agreement with the method of heredity of fused. Since those matings which produce a majority of fused among the females give a majority of wild-type among the biparental males (and the reverse), the males cannot be sex-reversed females.

It has also been suggested that half of the females resulting from matings of unrelated parents may be sex-reversed biparental males, in other words XX or YY females. This hypothesis fits the facts with respect to size of fraternities, ratio of males among biparentals and ratio of biparentals as correlated with relationship of parents, but it introduces the problem of explaining away the apparent absence of XX or YY females which should be equal to the XY females if parents are unrelated. The assumption that XX and YY females are present but sterile is contrary to the fact that high fertility is especially characteristic of females from outcrosses. The presumptive XX and YY females if fertile and heterozygous for fused would produce wild-type and fused in equal numbers among their biparental offspring whether males or females. XY females would also produce wild-type and fused in equal numbers if their biparental offspring were all females and equally viable whether XY or XX (or YY).

An absolute refutation of the hypothesis that some females may be XX or YY (sex-reversed males) must await further experimental tests. The truth of this hypothesis seems very doubtful, however, because there are no sex-intergrades between biparental males and females, because most females have been proved to be XY by sex-linkage of fused and because a supplementary hypothesis would have to be introduced to explain why XX or YY is female.

The reader may at this point refer to Figure 8 which expresses in round numbers the results of experiments carried out for the most part by C. H. Bostian.[1,2,3] As regards the experiment with unrelated parents, however, ratios of fused are merely presumptive for the experiment did not involve this factor.

Differential Maturation

It was clear at the time the theory of sex determination in Habrobracon presented above was advanced[14,15] that selective mortality alone was not adequate to account for the known facts when parents were outcrossed and that any possible scheme of sex-reversal necessitated so many subsidiary hypotheses as to be highly improbable. Selective fertilization was therefore suggested:

In the reduced egg there are four oötid nuclei, two being $1X$ + 1 set of autosomes

and two $1Y + 1$ set of autosomes. Normally three of these degenerate as polar nuclei in the peripheral cytoplasm. It is supposed that if an X-bearing sperm enters the egg there is selective fertilization, so that it fuses with a Y-bearing egg nucleus. Similarly if the sperm which has entered be Y-bearing, it will fuse with an X-bearing egg nucleus.

There is no discrimination against either type of sperm. Sperm from either type of male, X or Y, may enter the egg and fuse with a reduced egg nucleus.

Given a male of a certain type, X for example, all the sperm will be X-bearing and the selective action, which is one hundred per cent effective when parents are unrelated, involves discrimination against the reduced egg of similar type, the X-bearing, and in favor of the dissimilar, the Y-bearing, so that all syngamy is between X and Y, hetersyngamic or female-producing.

In the insect egg the four haploid sets of chromosomes resulting from the two oöcyte divisions remain in the egg cytoplasm. "Polar bodies" in the strict sense are not formed. In some insect species each of the four chromosome sets forms a nucleus and these may be numbered 1a, 1b, 2a and 2b in order from the more peripheral to the more central. Numbers 1a and 1b are the products of division of the first polar nucleus, while number 2a is the second polar nucleus. Number 2b is the definitive egg nucleus and is called the female pronucleus which will fuse with the male pronucleus derived from the sperm. Nuclei 1a, 1b, and 2a eventually disintegrate in the egg cytoplasm and indeed in many cases do so before they have taken nuclear form. In other words chromosome sets 1a, 1b, and 2a disintegrate as such without developing nuclear membranes.

At the time the theory of selective fertilization in Habrobracon was advanced it was not known whether there were four complete oötid nuclei present, although this was the view suggested. It was thought that selective action took place between the sperm nucleus and the egg nucleus of opposite types, selective syngamy, and that the two kinds of egg nuclei were completely formed. Unpublished cytological studies of B. R. Speicher have since shown that only one of the four haploid sets of chromosomes forms a complete nucleus and this is always the innermost set, 2b.

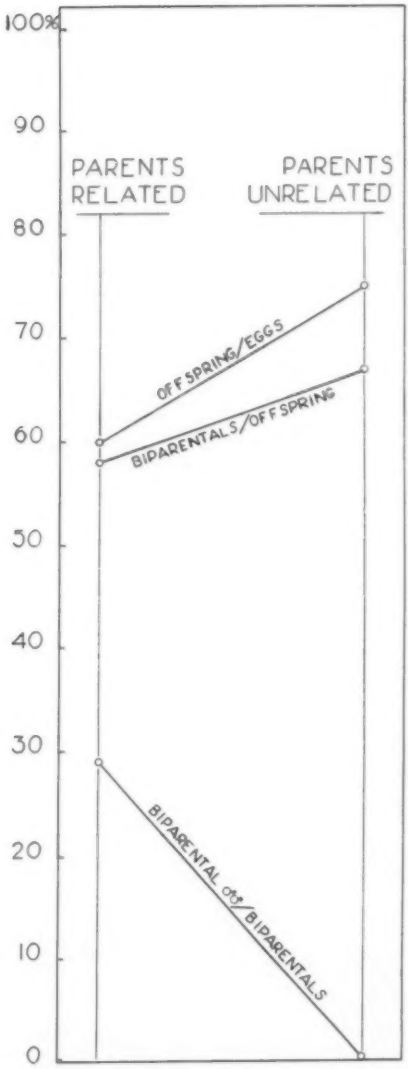
The concept of selective fertilization in Habrobracon, known by genetic tests to be selective syngamy between two different types of potential gametes within each egg, has therefore been further restricted to differential maturation. The sex chromosomes of the sperm nucleus must determine that the innermost chromosome set shall contain the opposite type of sex chromosome. The sex chromosomes have not as yet been identified cytologically.*

This hypothesis of differential maturation is the simplest explanation of the experimental results thus far obtained. The theory of sex-reversal of XX or YY individuals into females also fits the known facts, but necessitates supplementary hypotheses of sex determination for which there is no evidence.

Theoretical Considerations

Habrobracon may be compared with fishes in that crossing over in the sex chromosomes takes place between the sex determining region and genes not concerned with sex. The question has been asked: what would be expected if crossing over should occur within the sex determining region, between F and G for example? If F and G were by crossing over brought into the same chromosome, we might, on the complementary theory of sex determination suggested, expect a haploid female. Crossing over presumably does not occur and may possibly be prevented by an inversion of

*A recent paper by A. P. Guhl and R. L. Dozorceva, "A contribution to the knowledge of sex determination in Hymenoptera" (A. P. Guhl i R. L. Dozorceva: K. Vorprosu Opredeleniia pola u Hymenoptera. Doklady Akademii Nauk USSR 1934:522-526. Russian with English translation) presents cytological evidence that a Chalcidoid wasp, an endoparasite of cabbage butterflies, *Pteromalus puparum*, has, as the characteristic number in the female, ten chromosomes including four pairs with members equal and one pair with members markedly unequal both in size and shape. Two types of haploid males are shown corresponding with the two haploid sets expected from the female.



INFLUENCE OF RELATIONSHIP OF PARENTS ON VIABILITY OF OFFSPRING

Figure 7

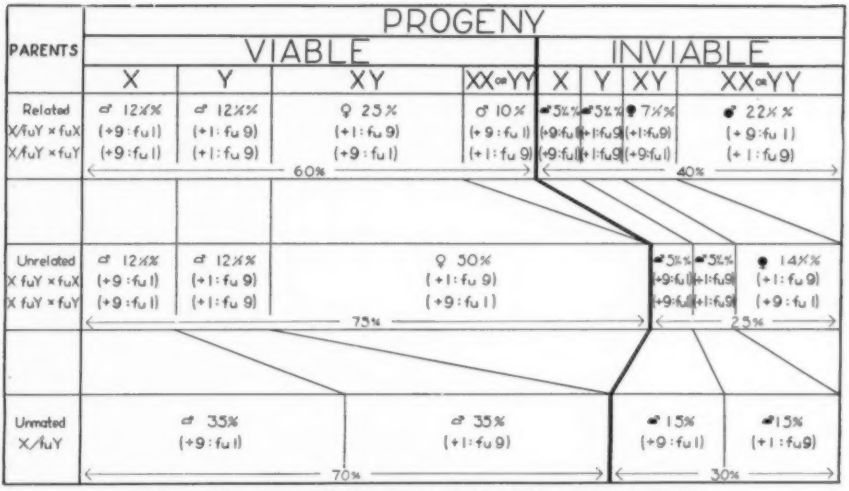
Influence of relationship of parents on percentages of eggs laid which develop into viable offspring (offspring/eggs); of biparentals among the viable offspring (biparentals/offspring), and of males among the biparentals (biparental ♂♂/biparentals).

the *Y*-region with respect to the *X*: the factors may be arranged in the order *F.g* in the *X* and *G.f* in the *Y*. But while the complementary theory necessitates that at least two genic differences are present, it must not be thought that there are necessarily only two. *F* may stand for a group of dominant genes in the *X*, *G* for a group of dominant genes in the *Y*. The presence of all or nearly all of these dominant genes is necessary for the production of a female.

As far as is known to the writer this is the first suggestion of complementary factors determining sex difference. Genotypically there are three sex types, if sex intergrades and mosaics are not considered: *X* (or *XX*) males and *Y* (or *YY*) males (which are phenotypically similar) and *XY* females. The doubling of the chromosome sets in the diploid males does not change the sex type. Genetic sex determination in other forms (excepting the gametophytes in plants) may be considered as the crossing of a heterozygous (or digametic) type to a recessive (or monogametic) type. The dominance tends to be all in one direction and it is the *Y* element which, "protected" by the *X* against the influence of natural selection, acts as the dominant. The recessive *XX* sex is in some groups the male in other groups the female.*

In Habrobracon with its two types of males, both *X* and *Y* chromosomes contain dominant as well as recessive factors and are equally subjected to natural selection. They cannot, therefore, degenerate by the accumulation of recessive lethals. Were it not for parthenogenesis, the complementary scheme might shift over into one of simple dominance, the crossing of a heterozygote with a recessive as in other forms. Thus a slight initial advantage of the *XX* males over the *YY* would mean that the *YY* would never be formed: all matings would be *XY* by *XX*. The *Y*-

*Muller[9] (1918) discussed "degenerative" changes taking place under the "protective" influence of enforced heterozygosis and summarized the phenomena with respect to the changes in the sex chromosomes.



RELATED AND UNRELATED MATINGS CONTRASTED

Figure 8

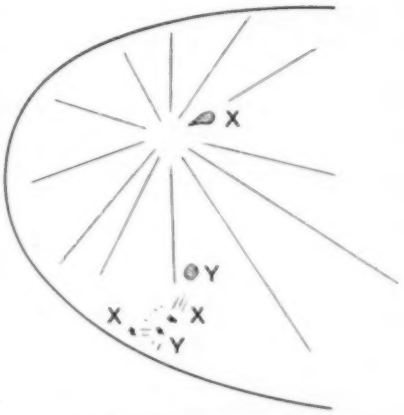
What may be expected if females of an inbred stock and heterozygous for fused-*Y* $\frac{X}{fuY}$ are bred unmated and if they are crossed with fused-*X* (*fuX*) and with fused-*Y* (*fuY*) males, both closely related and unrelated. The mothers were orange eyed so that biparental males could be distinguished from impaternate. The progeny are given as percentages of eggs developed into the various types (Viable) or as failing to develop (Inviable). The viable unfertilized eggs, *X* and *Y*, develop into haploid males which are wild-type, +, and fused, *fu*, in equal numbers. The crossover value between fused and *XY* is ten per cent with an excess of wild-type among the *X* males and of fused among the *Y* males. *X* and *Y* males are equally viable. Approximately thirty per cent of the eggs from unmated females fail to develop into adults, the greatest mortality being before hatching. About twenty-five per cent of the eggs laid by mated females develop into impaternate males, whether the females have been mated to related or to unrelated males. Presumably the ratio of unfertilized eggs which fail to hatch is the same whether the females have been mated or not and hence 3/7 of 25 per cent are counted as inviable unfertilized (5 5/14 per cent *X* and 5 5/14 per cent *Y*).

Twenty-five per cent of the eggs laid by females mated to related males develop into females, but only ten per cent develop into biparental males. If it be assumed that *XY* and *XX* or *YY* zygotes are formed with equal frequency but that the deficiency of biparental males is due to their greater mortality, the inviable fertilized will consist of more homeosyngamic combinations, *XX* or *YY* (22 1/7 per cent) than heterosyngamic, *XY* (7 1/7 per cent).

The cross with unrelated males shows that the hypothesis of differential mortality is not in itself adequate to explain the deficiency of biparental males in all cases. Fifty per cent of the eggs laid by females mated to unrelated males develop into females. (The percentage in Bostian's experiments was actually somewhat higher.) This is twice the total inviable, so that even if all the inviable were assumed to be homeosyngamic (*XX* or *YY*), they would be far from equalling the heterosyngamic (*XY*) actually maturing into females. Moreover since there seems to be no reason why homeosyngamic combinations from outcrosses should be more inviable than from crosses of related stocks and since no biparental males have ever been found among many thousands of biparentals from outcrosses, it is reasonable to suppose that all inviable fertilized are here heterosyngamic (females). If the proportion of inviable unfertilized calculated from the viable unfertilized (impaternate males) on the basis of progeny from unmated females, be deducted, the remainder will be the heterosyngamic inviable expected. These (14 2/7 per cent) prove to be in the same ratio to the adult females (50 per cent), as the calculated heterosyngamic inviable (7 1/7 per cent) are to the adult females (25 per cent) from the cross to related males. If we are correct in basing this calculation on the principle of random syngamy when parents are related, we must conclude that females from close breeding are as viable as females from outcrossing. How-

chromosome protected by the X might then accumulate recessive lethals and even disappear as it has in many types reproducing bisexually. With the type of parthenogensis occurring in Habrobracon, however, Y males and X males are produced with equal frequency regardless of the type of male with which mating occurs. There can then in the long run be no selection in favor of either type.

Nachtsheim's[10] suggestion with reference to differential maturation in the bee may be elaborated as follows. All males are haploid and of one type, $1X+1A$. Females are diploid and digametic as in Habrobracon, $1X+1Y+2A$. In the unfertilized egg the Y-chromosome passes into a polar nucleus along with one set of autosomes, so that a male, $1X+1A$, results. Fertilization causes a reversion of this type of maturation so that the X-chromosome passes out and the resulting zygote develops into a female, $1X+1Y+2A$. This scheme is consistent with modern ideas of genic balance on a ratio basis. Diploid males, $2X+2A$, might be formed as in Habrobracon by failure of differential maturation in the fertilized egg. Since all normal males are X the scheme of sex determination is not necessarily complementary as in Habrobracon, but may be compared with that in non-parthenogenetic forms. It involves the crossing of the heterozygous female to the recessive, in this case haploid, male. The Y-chromosome is "protected" from natural selection by differential maturation in the unfertilized egg. It is entirely possible that "Nachtsheim's" scheme may be the correct one for the honey-bee or may apply in certain other groups in which males are produced by haploid parthenogenesis. If this should prove to be the case in the bee, a sex-linked difference would be detectable in drone-broods. Except for crossovers, drones from a single heterozygous queen would be of one type only, the type determined by the X-linked factor.



MECHANISM OF DIFFERENTIAL MATURATION

Figure 9

Semi-diagrammatic view of the fertilized egg of Habrobracon shortly after being laid, shows the three polar nuclei, 1a, 1b and 2a, as groups of chromosomes situated near the ventral anterior surface. Groups 1b and 2a approach each other and form the polar fusion nucleus which degenerates after dividing once or twice. Only chromosome group 2b, the definitive reduced egg nucleus, becomes organized with a nuclear membrane and functions as a female pronucleus in fertilization. The tear-drop shaped sperm nucleus may be seen accompanied by astral rays. This soon becomes spherical, transforming itself into the male pronucleus.

Genetic evidence shows that if the sperm from an unrelated male carries an X-chromosome it exerts an influence in polar body formation so that the Y-chromosome of the egg remains in the central group, 2b, the group which will eventually function in fertilization. The reverse occurs if the sperm carries Y so that all fertilizations are heterogametic, XY, and female-producing.

In Habrobracon since X and Y males are produced with equal frequency, male broods fail to indicate sex-linkage. Broods from virgin females heterozygous for fused were bred extensively in linkage tests but failed to reveal sex-linkage since there is no differential maturation in the unfertilized egg.

Summary and General Conclusions

The inadequacy of the view that in bees and wasps the diploid chromosome

Legend to Figure 8—*Continued*

ever, since random syngamy, coupled with selective mortality, is inadequate to explain the results obtained from outcrossing, there may likewise be some limitation to random syngamy in close breeding. An excess of heterosyngamic combinations will fit the data if it be assumed that females from close breeding are less viable than females from outcrosses.

complex determines femaleness while the haploid determines maleness is discussed from the point of view of genic balance. A mendelian hypothesis of sex determination in the bee proposed by Castle in 1903, and also a theory suggested but not adopted by Nachtsheim in 1913, both involving differential maturation, are theoretically adequate although incorrect in certain details.

The discovery and development of the principle of sex determination in Habrobracon is outlined. The theory that there are two different types of impaternate males, each recessive to the female, was suggested by the occurrence of female-like structures in male mosaics. Search for sex-linkage was begun by testing twelve genes by crossing heterozygous females by single recessive males, usually the fathers of the females. Results were negative. Subsequent testing of the gene *fused* gave positive results showing about ten per cent crossing over with the sex determining region.

There are two types of biparental males resembling genetically the two impaternate types except that the chromosome complex is doubled. They are almost sterile and relatively low in viability. Females crossed with related males producing biparental sons are therefore less fecund than females crossed with unrelated males or virgin females.

Selective mortality explains in part the deviation from equality of biparental males and females expected on a mendelian basis.

In outcrosses, however, it is apparent that the relatively inviable biparental males are replaced by females, so that the biparental ratio in the adults is increased. The data may be explained by assuming that about one-half of the females from outcrosses are sex-reversed males. This theory necessitates subsidiary hypotheses of sex determination and introduces complications which are difficult to reconcile with the pedigrees.

The theory is advanced that in outcrosses the zygotic combinations from which males might be expected fail to occur, in other words genetic nuclei with similar sex chromosomes fail to undergo syngamy. This type of selective fertilization is brought about by differential maturation directed by the character of the sperm nucleus within the egg cytoplasm.

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SILVER GUINEA PIGS

A Recessive Color Variation

W. V. LAMBERT*

THE presence of white or white-tipped hairs in an otherwise pigmented coat is a character that has been observed in several species of mammals, usually as an age or a molting effect. In some species white hairs become so numerous in the pelage of animals of certain strains that they are designated as "silvers."

This character is probably best known in the silver fox, but in rabbits, also, it is a well-known color variation, being especially well developed in the variety known as the Argenté de champagne. In addition, it has been reported in two species of the mouse; in the house mouse (*Mus musculus*) by Hagedoorn[4] and by Dunn and Thigpen[3] independently, while in the deer mouse (*Peromyscus maniculatus gambelii*) Sumner[5] has described a somewhat similar character which he called "grizzled."

Origin and Description

This character was first observed in a few closely related individuals in 1928. From these foundation animals, all of which showed a low degree of silvering, our present stock has descended. The fact that silvers had not been observed previously in this colony since its establishment in 1923 would indicate that the character was of rather recent origin.

Silver guinea pigs, in common with individuals of this color-phase in other species, show much variability in degree of silvering. For convenience in description all silver animals have been classified, depending upon the degree of silvering shown, into one of three grades, namely high, medium or low. Figure 10 shows the respective grades of silvering (*A-D*), while Figure 10*E* shows a non-silvered black guinea pig.

In the high grade silvers (see Figure 10*A*) much of the black hair over the posterior region of the back and sides and to a lesser extent on the head and belly is replaced by white hair. In silvers, even of the low grades, the presence of white hairs just above and in front of the ears is very characteristic, though the white hairs are more numerous here in high than in low grade silvers. In low grade silvers the white hairs are confined largely to the posterior region of the back and are much fewer in number (see Figure 10*D*). The white hairs appear first on the posterior region of the back, the area in which they subsequently become most numerous.

In the silvers no white hairs are present at birth and forty days is the youngest age at which white hairs have been observed in the coat. In most individuals they are noted first between the ages of three and four months. After this age white hairs appear rapidly and, in general, one is able to classify the degree of silvering accurately when an animal is six months old. However, a slow but gradual increase in the degree of silvering takes place in some animals until they are two years old.

Microscopic examination of glycerin preparations of the white hairs in the silvers shows them to be without pigment throughout their entire length. In this respect the character differs somewhat from that of the rabbit and fox in which, according to Castle[2] and Ashbrook,[1] the condition is characterized by the presence of white, white-tipped and banded hairs. In the house mouse Dunn and Thigpen observed both types of hair, while Sumner reported that the

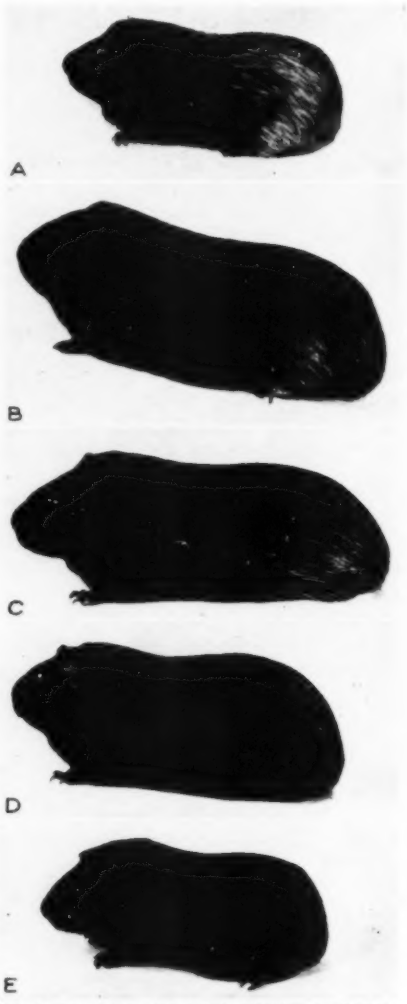
* Paper No. 68. Department of Genetics, Iowa State College, Ames, Iowa.

grizzled character in the deer mouse resulted from the replacement of colored hairs by white hairs in certain parts of the coat.

Inheritance of Silvering

Silvering is a recessive variation of the normal or non-silvered pattern. However, dominance of the non-silvered condition is not complete, for some individuals heterozygous for silvering develop a few white hairs in their coat. The recessive nature of silvering is evident from the results of outcrosses of silvers with unrelated non-silvers and, also, from the matings of silvers with silvers. From six different matings of the former type fourteen offspring were produced, in twelve of which no white hairs were observed, while in two animals a trace of silver was evident in late molts. Twelve different matings of silvers with silvers produced thirty-nine progeny. Thirty-eight of these showed enough white hair in their coats to be classed as silvers while one individual showed only a trace of silvering. The latter individual came from a low-grade silver dam in which the silvering was rather late in development. The summarized results of these matings are shown in Table I (*A* and *B*).

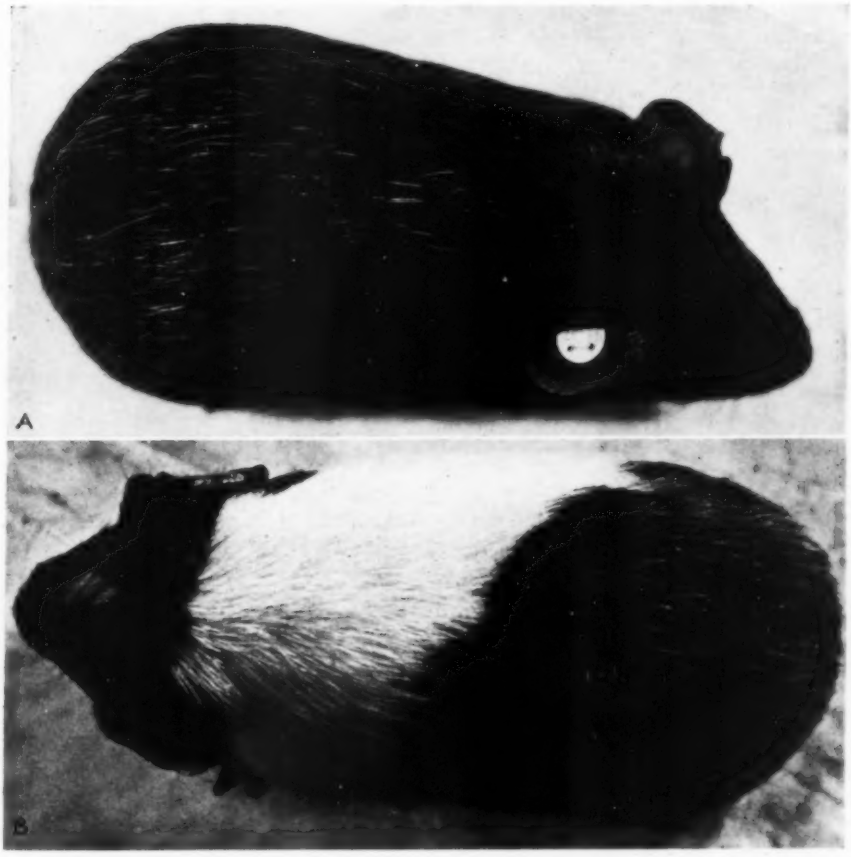
In order to determine something of the mode of inheritance of silvering, reciprocal matings of heterozygous individuals with silvers and heterozygotes with heterozygotes were made. The respective results of these two types of matings are shown in Table I (*C* and *D*). From the results it would appear that the silver character is dependent upon one major recessive factor. From matings of the first type a total of sixty-three individuals, showing no silvering or only a trace, and fifty-eight animals having enough white hairs to be classed as silvers, were produced. From the matings of heterozygotes *inter se* twenty-four non-silvers or with a trace of silver, and six silvers were obtained. The results of both types of matings are in substantial agreement with a monogenic mode of inheritance.



SILVER AND BLACK GUINEA PIGS

Figure 10

Silvering is a new recessive coat color variation in the guinea pig. The character, which is due to the development of entire white hairs among the pigmented hairs of the coat, is not evident in the pelage of the new born, but develops as an age or molting effect. The range of variation of the character in mature animals is shown above. *A*, a high grade silver; *B-C*, medium grade; and *D*, a low grade silver; *E*, a black non-silver. White hairs first appear in the coat when an animal is from two to three months old.



DORSAL VIEWS OF HIGH-GRADE SILVERS

Figure 11

A is a self black individual, heterozygous for the spotting gene (genotype *Ss*), while *B* shows an individual with the genotype *ss*. The character is developed to an equal degree in these two genotypes and also in animals of genotype *SS*. These two guinea pigs were litter mates and approximately six months old when the pictures were taken.

However, from the variation in the degree of silvering it is apparent that the character must be influenced by one or more modifying genes. Because of the limited data and the difficulty in classification it seems useless at this time to formulate a definite factorial explanation of the mode of inheritance of silvering. In the amount of variability shown, the silver character in the guinea pig appears to be much like the similar character in the fox, mouse and rabbit. Some individuals develop many white hairs in their coat whereas others develop relatively few. This fact makes classification difficult, but at the age of six months it is possible to determine quite accurately whether an animal should be classed as silver or non-silver. To determine the exact degree of silvering is more difficult, for some individuals continue to develop

white hairs in their pelage until they reach about two years of age.

There is a tendency for high grade silvers to produce silvers of a higher grade than do low grade animals. This is evident from the results of the matings of silvers with silvers shown below.*

Results are not yet available from *inter se* matings of the lower grade silvers.

It is apparent from the matings of heterozygous (non-silver) males with silver females, and from the inter-mating of heterozygotes, that the gene for silvering is not sex-linked. Silver females were produced in both types of matings, thus precluding a sex-linked mode of inheritance.

There is, likewise, no evidence that the character is sex-limited, it being equally well developed in males and females.

Data from some of the matings of heterozygous animals with silvers indicate that the silver gene is inherited independently of the factor for white spotting (*s*) and of the genes in the extension series (*E, eP, e*). However, a larger number of progeny from these matings is necessary before final conclusions can be drawn.

Degree of Silvering and Age of Appearance

As previously mentioned, the silver character is not evident in the individual at birth but develops only in later molts. To determine the approximate age at which white hairs first appear in the coat all individuals were examined approximately at monthly intervals, and the degree of silvering recorded.

While considerable variation in age at first appearance of silvering is observed in all groups, high grade silver animals develop white hairs in their fur at the youngest age, followed in turn by medium and low grade silvers. The age at which white hairs first appeared in animals showing a trace of silvering was greatest, although the likelihood of overlooking a few white hairs in the coat no doubt has increased to some extent the age at first appearance of white hairs in this group. The average age at which white hairs were first observed in the various groups was as follows: high grade, 90 days; medium, 110 days; low, 135 days; and for animals showing a trace of silvering, 170 days. No difference in age at first appearance of silvering was noted between males and females.

Discussion

Silvering has been observed in animals of several different color phases: self blacks (*e*), black and reds (*ep*), reds (*ee*), in intense (*C*), dilutes (*Cd*), and in self color (*S*) and white spotted (*ss*). No difference in degree of silvering or in the age at which the white hairs appeared thus far has been noted for the various colors. Individuals of genotypes *SS, Ss* or *ss* show silvering to about an equal degree thus indicating there is no influence of the white spotting gene upon the expression of the silver character. Dorsal views of two high-grade silvers, one of genotype *Ss* and the other of genotype *ss,* are shown in Figure 11. The spotting gene, however, is undesirable in the silver stock as an occasional *ss* animal has such extensive white areas that it becomes impossible to determine the degree of silvering.

Because of the late development of white hairs in the fur of silver animals, it is obvious that this is an unsatisfactory character for genetic studies since many animals die before the character can be definitely determined. From the physiological standpoint, how-

*Type of mating	Number of progeny in various grades			
	High	Medium	Low	Trace
High × High	7	1	1	
High × Medium	11	6	3	
High × Low	4	3	2	1

ever, it is an interesting character. Why, for instance, does the hair follicle in silvers change with age so that it loses its pigment secreting power, and, furthermore, why should this effect be limited only to certain follicles? In addition, why should this change be confined largely to certain regions of the body, as is apparent from Figure 10*A*? It appears as if there are various thresholds for pigment production and that this threshold changes with age. Castle[2] has suggested that the silver condition in rabbits may be related physiologically to the whitening of the coat which takes place in winter in the Northern Varying Hare of the northeastern part of the United States. In the latter case the change seemingly is evoked by a lower temperature. In the guinea pig the temperature factor would not appear to be the causal agent of the failure of the hair follicle to produce pigment as the white hairs appear comparatively late in life and, as previously mentioned, in silvers the number of such hairs gradually increases until an individual reaches the age of about two years.

In many respects silvering in the guinea pig is similar to the graying observed in man. It develops comparatively late, gradually increases in degree and exhibits much variability in age at development and in total degree of development in different individuals. A somewhat similar character occurs in horses. According to Wright[6] "grays generally show little or no white when born, but gradually become whiter as they grow older, white appearing early especially on the head." The gray character in the horse acts as a dominant over self or non-gray, and, from the meagre evidence available, is possibly governed by one dominant factor.

Table I. A summary of all crosses in which silver animals or animals heterozygous for the silver character were used.

Type of mating	Progeny				
	Non-silvered	Trace of silvering*	Silvered (by grades) High	Medium	Low
(A) Silvered × Non-silvered (outcross)	18	3	0	0	0
(B) Silver × Silver	0	1**	22	10	6
(C) Silver × Heterozygous	35	28	13	21	24
(D) Heterozygous × Heterozygous	17	7	1	5	0

* Showing only a few white hairs in their coat.
** This individual was from a low grade silver dam.

Summary

1. A new recessive color variation, called "silver," is described in the guinea pig. In many respects it resembles the similar character in the fox, rabbit and mouse.

2. The variant is characterized by the development of totally white hairs in a pigmented coat. The white hairs do not appear in the first coat, but only in the second or later molts. Some relation between degree of silvering and age at development of white hairs in the fur exists, the higher grade silvers showing white hairs in their coat at an earlier age than low grade silvers.

3. Breeding tests suggest that the inheritance of silvering is governed by one major recessive gene, but that the expression of the character (degree of silvering) is influenced by plus and minus modifiers. The silver gene is not sex-linked and is independently inherited from the white spotting gene and from the factors of the extension series.

4. Silvers have been produced in several different color phases but the character seems to be equally well developed in all of the color phases so far observed.

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SEXUAL PHOTOPERIODICITY IN ANIMALS

Comments on a recent discussion of the subject by Dr. S. Zuckerman

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IN his book "Functional Affinities of Man, Monkeys and Apes," published in 1933 by Harcourt, Brace & Co. and reviewed in this JOURNAL, May, 1935, Dr. Zuckerman, without citing the papers of the authors referred to, so that his readers may refer to them and judge for themselves how far he gives their ideas, says (page 31):

> The extent of an animal's breeding activity is affected by many external factors (e. g., food and warmth), and some writers, on the basis of experiments on starlings, ferrets, and field mice, are inclined to generalize that an animal's breeding potentialities vary directly with the amount of light to which it is exposed. There can be no doubt that this is an unsound and unjustifiable generalization, for a critical survey of the evidence points clearly to the conclusion that factors inherent in themselves, and not immediate external conditions, determine whether animals have *absolutely* restricted breeding seasons, or whether they are capable of breeding throughout the year. Thus, the same conditions of light do not stimulate the baboons and lemurs in the London Zoological Gardens to breed at the same time of year. Fundamentally, an animal's breeding habit [sic] is part of its inherited physiological constitution.

That this gives a false impression of the conclusions and very tentative and limited generalizations of the "writers" alluded to will be evident enough if their papers are read. Bissonnette[6–10, 12–15] and Bissonnette and Wadlund[17–19] are probably the ones referred to as experimenting with starlings; Bissonnette[11, 13, 16], Hill and Parkes[22–25], McPhail[27] (1933), Allanson, Rowlands and Parkes[2], Marshall and Bowden[26] and others have studied ferrets; and Baker and Ranson[3–5] did relevant work on field mice. In none of their published papers has the writer been able to find even implications that they "are inclined to generalize that an animal's breeding *potentialities* vary" in any way in respect to environmental conditions, let alone "directly with the amount of light to which it is exposed." They say nothing about modifying the *potentialities*, and appear to take them for granted as specific genetic characters. They may perhaps be said to indicate that they feel that, in *some* animals, the time of year at which their restricted breeding seasons occur may be changed by altering the light cycles to which the animals are exposed. They say nothing at all about animals capable of breeding throughout the year, on which their work to date does not bear.

If "a critical survey" of their papers had been made, probably they would not have been misinterpreted as was done in the above quotation.

More than that, the conclusion given in the last sentence, that "fundamentally, an animal's breeding *habit* is part of its inherited physiological constitution" is not based on critical analysis. The writer agrees that an animal's breeding or sexual *potentialities* are "part of its inherited physiological constitution"; but its *habits* are the result of the interaction of these potentialities with its environment, which may, and evidently sometimes does, act to inhibit or to release or stimulate the manifestation of the inherent potentialities or tendencies in one direction or another. Light cycles for starlings, ferrets, and field mice, without peradventure, do modify the breeding *habits* of these animals in some cases. This is clearly shown for ferrets by the change over of their oestrus or breeding period, when they are taken to Australasia, from the March-August months of the northern hemisphere to the August-January or September-February months in the southern hemisphere (data kindly furnished by Dr. Zuckerman, himself, 1932), and by experimental modifica-

tion of the light cycles carried out in the north by the authors cited above.

That this phenomenon is not confined to the animals mentioned above is evident from studies of Shapiro and Shapiro[31] and Zwarenstein and Shapiro[32] on *Xenopus laevis,* the clawed toad of South Africa; from those of Cole[20] on doves in which he obtained viable eggs in the winter quiescent period of the mourning dove; from those of Miyazaki[28] with *Zosterops palepebrosa japonica,* the Mejiro of Japan, which he induced to come into breeding condition three times in a single year in place of once by the "yogai",* used for many years by bird fanciers in Japan; from those of Allanson and Deanesly[1] with hedgehogs, in which they found the sexual cycle influenced by light among other factors.

That light time and intensity are not the only factors modifying or conditioning the sexual cycles of the above mentioned animals is quite evident from the published papers of the above cited writers, and they make much of the facts. It is also shown by other writers, cited by them, that, in some animals, sexual activity is induced upon a falling instead of an increasing light period, e. g., sheep, or is little affected if at all by alteration of light cycles or absence of light (Hill and Parkes[22]; Moore, Simmons, Wells, Zalesky, and Nelson[30]; Dempsey, Myers, Young, and Jennison[21]; with ferrets, spermophile ground squirrels, and guinea pigs, respectively).

The writers given above have been very careful not to generalize by analogy from one species to another. Their studies show that in some animals the times of oestrus or sexual activity and of anoestrus or quiescence may be changed and breeding *habits* modified by addition of artificial light to normal days in autumn and winter, when normally the animals studied have a long anoestrus or quiescent period. They also show that, while the animals' specific inherent inherited sexual potentialities do not change, the manifestations of those potentialities, to become sexually active and breed at any time of year, are conditioned and controllable by the timing and type of light cycles to which the animals are exposed, in different degrees in different species of animals. That, in some, they are highly dependent upon cycles of daily light periods and little affected by many other environmental factors, while in others the light factor is of little or perhaps no importance in relation to other factors, is the general conclusion to be drawn from the studies above cited. That this is the opinion of the "writers" referred to by Dr. Zuckerman can be seen by a critical reading of their published papers. It is hoped in a later issue of this JOURNAL that it may be possible to present a more general résumé of this subject.

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✦

MORE ADVICE FOR PARENTS

THIS little volume* is very readable and interesting. I was frankly prejudiced against it, because I read in the reviews of it that it had the psychoanalytic outlook, but I found the psychoanalytic outlook tempered by so much of common sense that it was most refreshing. Dr. de Kok is quite unlike the modern psychologists whose attempt to understand children consists of treating them "as if they were adults, never kissing them, but shaking hands with them, patting them on the head if they have done a job well." She evidently believes that loving her children, playing with them, reading to them, assisting them is good for the social development of the children, and unlike some of the modern teaching which urges that children not be repressed so that they may never develop fear complexes, she actually believes in making them mind, and in calling in moderate physical punishment to assist her in making clear to them that certain things must not be done.

In dealing with children's problems, she gives many experiences of her own with her small son and daughter. These should prove helpful to the mother who is intelligent, but who has not the background of training to enable her to handle situations in the most satisfactory manner. Unfortunately, many mothers have not the mentality to understand and put into effect a rational treatment of children, and so for them the book is useless, and many intelligent mothers have evolved their own methods. For the group having the intelligence without the training, the book should prove of value. One of the difficulties is that no two children ask questions alike, or react to circumstances in the same manner. Hence the most that such a book can do is to give general principles, and hope that the individual mothers will work out their own technique. The numerous illustrations of her own specific handling of situations will help the mother to see how they might be handled without giving her instructions as to the management of a similar situation with her own child, for her own child will probably ask its questions, or behave, in such an individualistic way that Dr. de Kok's methods and answers are not strictly applicable. Nevertheless her sensible and wholesome outlook will probably help other mothers to orient themselves more adequately to the serious business of raising a family.

Madge Thurlow Macklin.

*de Kok, Winifred. *Guiding Your Child Through the Formative Years.* Emerson Books, Inc., New York. 1935. $2.00.

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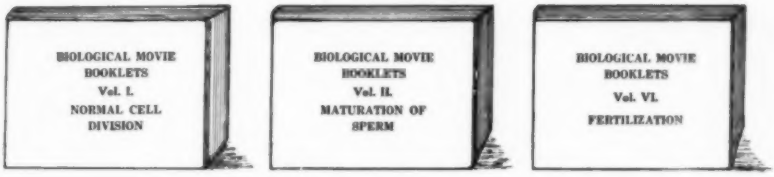

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